

SCLEROSIS OF THE CORNU AMMONIS IN EPILEPSY.

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(Continued from April Number.)

Case 7.—E. R., a negro girl, aged 13, died July 6, 1891, after a residence of two years and five months in the Arkansas asylum, of pulmonary tuberculosis. When received, her right leg was flexed, by contraction of the hamstring muscles, to such an extent as to make walking very difficult, and the contracture increased during her stay until the limb could not be extended much beyond a right angle. She was imbecile to a considerable extent, but could talk very well, was very observant about many things and never gave evidence of any defect of vision or other anomaly of sensibility, general or special. She was stated to have been subject, before admission, to convulsive seizures occurring about once a month, but was only known to have three or four while under treatment.

At the autopsy, there was found to be great atrophy of both occipital lobes of the cerebrum. The supramarginal gyri were small, but apparently healthy; the angular and the occipital convolutions of the external surface of both hemispheres formed an irregularly puckered mass, in which the convolutions could not be distinguished. The same was the case with the cuneus, most of the præcuneus and the occipital part of the occipito-temporal convolutions of the right hemisphere. In the left, the paracentral lobule was much atrophied; the place of the præcuneus was occupied by a cyst containing about two drachms of fluid, and the cuneus reduced to a translucent membrane with some streaks of cerebral matter running over it. The posterior cornua of both lateral ventricles were much dilated, and the cornua ammonis were unduly small—the left distinctly smaller and harder than the right. On median section of the cerebellum, marked sclerosis of the white matter of the arbor vitæ was evident, and the right corpus dentatum cerebelli was also sclerotic.

Microscopical examination of the right hippocampus shows interruption of the stratum pyramidale just before its entrance into the nucleus fasciæ dentatæ. The latter contains pretty numerous characteristic cells, but is irregular in shape, and of not more than half the normal size. The stratum granulosum is almost entirely wanting in some places. In the left, the stratum pyramidale and nucleus fasciæ dentatæ are both almost deficient in characteristic cells, except near their junction, and the nucleus is very small and irregular in shape. The stratum granulosum is pretty well preserved.

In the occipital lobes, the degree of atrophy varies from a condition in which the gray matter seems pretty well preserved, but the medullary portions of the convolutions much shrunk, to a complete absence of nerve-cells and fibres. The atrophied portions are occupied by neuroglia, with but few and inconspicuous spider cells. The histological appearances of the atrophied portions here and in the cornua ammonis are identical.

Case 8.—G. M., white, male, aged 34 at death. Inmate of the Arkansas asylum for ten weeks. The only pertinent information obtained as to his previous history was the statement that he had at some time suffered from "over-heat," and had been subject to convulsions for fourteen years. His mind was inactive, but he was capable of coherent conversation and conformed readily to the regulations of the ward. He had rather frequent attacks of grand mal, controlled to a considerable extent by bromide. There was no paralysis, and no aphasia. He died August 23, 1893, of dysentery.

The brain weighed forty-four ounces. A patch of atrophied and sclerotic tissue extended the whole length of the right first temporal convolution, involving the greater part of the supra-marginal gyrus, and part of the island of Reil. In parts there was a brownish discoloration of the cortex; elsewhere, although shrunk and abnormally hard, it retained its normal color. In the left hemisphere, a similar patch, nearly two inches in the horizontal and one in the vertical direction, was found in the superior and inferior parietal lobules, and another occupying the tip of the temporal lobe and extending over the anterior two-thirds of the first temporal gyrus. The cornu ammonis of that side was also much smaller and firmer than that of the right, which seemed normal.

Microscopically, the atrophied portions of the temporal and parietal regions are made up of a belt of neuroglia fibres, in some parts dense, in others loose, with pretty numerous nuclei, but few large spider cells. Similar tissue occupies the stratum pyramidale, stratum lacunosum and nucleus fasciæ dentatæ of the left cornu ammonis, dense in the nucleus and very loose in the other portions. The nucleus is almost destitute of nerve-cells, and the external half of the stratum granulosum is almost entirely wanting.

Case 9.—R. M., white, female, aged 43 at death. Inmate of the Arkansas asylum for nearly seven years. She was stated to have suffered from puerperal convulsions in 1876 and to have been insane ever since. The date of commencement of her epilepsy was not stated. At the time of her admission she was entirely blind, denying any perception of light. Ophthalmoscopic examination by myself and Dr. T. E. Murrell, failed to disclose any lesion sufficient to account for the blindness. She had rather infrequent but severe convulsions. Mentally, at the time of admission, she was considerably demented, but cleanly in her habits and capable of answering simple questions. Her dementia gradually increased, and in July, 1891, she was noticed to have some paresis of the legs. The knee-jerks were normal or nearly so, and no evidence of anæsthesia could be found. The paralytic trouble increased rapidly; by September 1 she was unable to walk unaided. and by December 10 of the same year she could not stand alone. In walking with support she raised the feet high and set them down flat. In the course of the following year she became entirely helpless, as far as the lower extremities were concerned, and a moderate talipes varus of the right foot developed. She still seemed to have full control of her hands. Mentally, she was almost completely demented, extremely irritable, and disposed to resist everything that was attempted with her. On this account it was difficult to make an ophthalmoscopic examination, but Dr. F. Vinsonhale succeeded in getting a view of the optic disc, and reported white atrophy of the optic nerve. She died March 14, 1894, of gangrene of the lung.

The brain was evidently considerably atrophied, and there was a large amount of sub-arachnoid fluid. It weighed $32\frac{1}{2}$ ounces. The right occipital lobe presented a large, sunken patch, of brownish color, occupying almost the whole of its external surface, and extend-

ing over its upper border to the internal surface, involving the anterior third of the cuneus, the whole præcuneus, and extending in a narrow strip for about one and a half inches along the sulcus calloso-marginalis. A similar patch occupied nearly the whole surface of the left occipital lobe. The disease extended through the white matter to the posterior cornua of the lateral ventricles. There was a similar, very superficial alteration of the genu of the corpus callosum. The right cornu ammonis seemed harder than normal, but not very much diminished in size; the left was very hard and small. The lateral ventricles were much dilated; their ependym smooth. The optic thalami seemed smaller than normal; the left somewhat flattened.

Microscopically, in the right cornu ammonis, the stratum pyramidale is destitute of nerve-cells, except in a small portion just before its entrance into the nucleus fasciæ dentatæ, which latter, although of nearly normal size, is entirely barren of nerve-cells. The stratum granulosum is well developed. In the region of the lamina medullaris interna is a very loose reticulum of neuroglia. The left cornu ammonis can be described in almost identical terms, except that the nucleus fasciæ dentatæ is much smaller than in the right and contains a very few nerve-cells. Sections of the diseased portion of the left occipital lobe show it to consist of a belt of neuroglia fibres, in some parts dense, in others loose, abundantly vascular, and containing numerous spider cells. Through a considerable extent of its surface a thin layer of cerebral substance seems to have been separated from the subjacent tissue, with which it is only connected by a loose net work, consisting mainly of tortuous blood vessels. The structure of the diseased portion in this region differs from that of the atrophied parts of the hippocampus mainly in the greater abundance of spider cells.

In the foregoing cases it seems to me more reasonable to suppose that the sclerosis of the cornu ammonis is due to the same causes as the other lesions than that it results indirectly from them, as an effect of the convulsions. This, however, is not a matter capable of demonstration, and each one must form his own opinion from the evidence, which I have tried to present fairly.

I have found organic lesions, not accompanied by disease of this convolution, in four other cases of epilepsy. In one, a case of infantile hemiplegia, the left hemisphere,

which was not enlarged, forming a mere thin shell over the enormously enlarged lateral ventricle. In another, there was a cyst in the posterior part of the first left frontal convolution. The third had a small focus of softening in the posterior part of the Island of Reil, and another in the posterior part of the first temporal convolution, both on the left side. The fourth, a case of tumor involving the temporal lobe, will be detailed later.

To discuss the bearing of some of these cases on the theory of cerebral localization is aside from my present purpose.

Whatever may be thought of the relation between the disease of the cornu ammonis and of other parts of the brain in the foregoing cases, I presume it will not be questioned that the anatomical changes, taken together, were, probably in all of them, the cause, rather than the effect, of the convulsions—that they were cases of “organic” as distinguished from “essential” or “idiopathic” epilepsy. All but one of them presented during life symptoms pointing to cerebral disease, other than convulsions. The following eleven cases, on the other hand, showed no symptoms by which they could be distinguished from others in which no gross anatomical change could be discovered. All of them were cases of chronic epilepsy, subject to more or less frequent attacks of *haut mal*. Whether the lesions found are due to the same cause in the two sets of cases may be open to question, but they are, anatomically, to all intents and purposes, the same.

Case 10.—R. H., white, male, aged 26 at death. Said to have been insane four years at the time of his admission to Arkansas asylum, March 11, 1889. No information furnished as to the duration of his epilepsy; the only statement made in regard to it was that he was subject to fits “after drinking whiskey.” Died Sept. 6, 1890, of diarrhœa.

At the autopsy the only lesion discovered was in the right cornu ammonis, which was abnormally small and hard.

Microscopically, the nerve-cells of the stratum pyramidale and nucleus fasciæ dentatæ of this convolution were found to be almost entirely lacking and these portions much reduced in size. The stratum granulosum was well developed. The left hippocampus and other convolutions, so far as examined, presented no important alterations.

Case 11.—P. V., white, male, aged 25 at death; resident of the Arkansas asylum for five years. Duration of epilepsy not stated; insanity said to have shown itself at twelve years of age. He was extremely demented, restless and excitable. Died Aug. 21, 1891, of epilepsy.

At the autopsy, the brain appeared healthy with the exception of the left cornu ammonis, which was sclerotic.

Microscopically, in this convolution, the stratum pyramidale is obliterated for a short distance near its origin, by the union of two bands of sclerotic tissue, starting, one above, the other below the gray matter of the subiculum. During the remainder of its course, it is represented by a small number of irregularly placed cells. The nucleus fasciæ dentatæ is almost entirely deficient in nerve-cells, and the stratum granulosum much thinned. In the other convolutions examined, some spider cells were found in the white matter, and, in portions, some proliferation of neuroglia in the most superficial layer of the gray matter.

Case 12.—C. C., mulatto, male, aged 40 at death; inmate of the Arkansas asylum for five years. Duration of epilepsy unknown. Subject to rather infrequent but severe convulsions. Moderately demented; worked pretty steadily at rough labor. Death occurred Sept. 5, 1891, in status epilepticus.

The only cerebral lesion discovered at autopsy was atrophy of the right cornu ammonis.

Microscopical examination of the affected convolution showed only few and scattered nerve-cells in the stratum pyramidale and nucleus fasciæ dentatæ, which took the carmine stain more deeply than the surrounding tissues. The stratum granulosum was well preserved. In other convolutions examined, slight proliferation of neuroglia was found in the most superficial portions and in the white substance.

Case 13.—M. C., white, male, aged 29 at death. Said to have been subject to epilepsy since four years of age. He was extremely demented and suffered from frequent and severe convulsions, in one of which he died Sept. 12, 1891.

Atrophy of right cornu ammonis was found post-mortem.

Microscopically, the stratum pyramidale is destitute of nerve-cells, except for a short distance just before its entrance into the nucleus fasciæ dentatæ, which is greatly

reduced in size and contains scarcely any nerve-cells. Stratum granulosum is almost destroyed in its inner and lower portions. In the other convolutions the changes were of the same kind as described in the preceding two cases.

Case 14.—B. J., mulatto, male, aged 32 at death. Inmate of the Arkansas asylum for ten months. Said to have become epileptic at eight years of age. Extremely demented and filthy in his habits. Attacks of grand mal moderately frequent. Died March 25, 1893, of pyelonephritis.

There was no appearance of general atrophy of the brain, which weighed forty-four ounces. The right hippocampus major was atrophied in all its dimensions—less than half as broad as left. The type of convolution was somewhat irregular; the interparietal and first temporal fissures were bridged on both sides.

Microscopically, in the right cornu ammonis, the stratum pyramidale and the nucleus fasciæ dentatæ are both almost entirely destitute of nerve-cells, and the stratum granulosum absent in the greater part of its extent. In specimens stained by Pal's method, the deep layer of the alveus and the medullated fibres of the hilum of the fascia dentata are much smaller than usual.

The left hippocampus seems normal, with the exception of an appearance of slight sclerosis for a short distance in the stratum pyramidale.

Case 15.—C. C., white, female, aged 38 at death; resident of the Arkansas asylum for nine years. Duration of epilepsy not stated. She was very much demented, violent, and at times manifested katatonic symptoms, standing for hours together in constrained positions, making peculiar gestures, etc. Died March 15, 1893, of cancer of uterus.

The brain was very small, weighing, with the membranes, only 35 ounces, but seemed of normal color and consistency. Right cornu ammonis much smaller and harder than the left.

Microscopically, in the right hippocampus, the nerve-cells of the stratum pyramidale are absent, except in a small portion just before its entrance into the nucleus fasciæ dentatæ, which is also nearly destitute of nerve-cells. The stratum granulosum is greatly thinned in a large part of its extent. The internal layer of the alveus is much

thinner than normal, but there seems to be no great deficiency of nerve-fibres in the nucleus. The other cornu ammonis and other convolutions examined present no very noteworthy anomaly.

Case 16.—S. C., white, male, aged 42 at death. Duration of epilepsy not stated. Had worked as a printer and, during the time of his residence in the asylum was always a pleasant, inoffensive, moderately demented man. Convulsions of moderate severity, not very frequent. Died Nov. 7, 1893, after having been a patient for seven years, of gangrene of lung.

At the autopsy, the brain, which weighed $40\frac{1}{2}$ ounces, seemed healthy, with the exception of the left cornu ammonis, which was much atrophied, and of almost cartilaginous hardness.

Microscopically, there is almost entire deficiency of nerve-cells in the stratum pyramidale and nucleus fasciæ dentatæ, and the stratum granulosum is atrophied to a great extent. The internal layer of the alveus is almost entirely wanting, but medullated fibres are fairly abundant in the atrophied nucleus. In other convolutions, a thickening of the neuroglia on the surface and some development of spider cells in the white matter are seen.

Case 17.—T. McL., white, male, of no occupation, said to have been epileptic since six years of age. Resident of the Arkansas asylum for seven years. He was subject to rather frequent convulsions; his mental condition was characterized by quiet dementia. Died Feb. 9, 1894, of epileptic exhaustion.

At the autopsy, the brain appeared healthy with the exception of the cornua ammonis, which were both abnormally small and hard throughout. In both hippocampi, microscopical examination shows deficiency of nerve-cells in stratum pyramidale and nucleus fasciæ dentatæ. In the right, a few are found for a short distance before the entrance of the former into the latter. The strata granulosa are well preserved in the greater part of their extent. Elsewhere, nothing distinctly abnormal is noticed except slight superficial thickening of neuroglia in some places.

Case 18.—V. T., white, female, aged 20 at death, resident of asylum two years. Said to have been epileptic from childhood, and to have shown mental derangement when seven or eight years old. Much demented, violent and erotic. Died Dec. 15, 1893, of diarrhœa.

Although she was considerably above average size, the brain weighed only $36\frac{1}{2}$ ounces. The right cornu ammonis was much smaller and harder than the left.

Microscopically, in the right cornu ammonis, the stratum pyramidale is fairly well preserved for a short distance before its entrance into the nucleus fasciæ dentatæ; with that exception, it is destitute of nerve-cells, and but few are to be found in the shrunken nucleus. The stratum pyramidale is much thinned, and so is the inner layer of the alveus. Medullated fibres are not very numerous in the nucleus. In the left cornu ammonis, and in other convolutions examined, little that is abnormal can be found except spider cells in moderate numbers.

Case 19.—E. O., a girl, aged 20, died Sept. 25, 1896, in Danvers Lunatic Hospital, of tuberculosis. She was said to have been epileptic since eight years of age, and was extremely demented. Convulsions were frequent and severe.

At the autopsy, apart from very soft consistency, probably due to post-mortem change, no lesion of the brain was found except sclerosis of the left cornu ammonis, which was much smaller than the right and abnormally hard.

Microscopically, the right cornu ammonis appeared entirely healthy. In the left, the nerve-cells of the stratum pyramidale were almost entirely wanting, and only a few scattering ones remained in the nucleus fasciæ dentatæ. Spider cells, in the atrophied portions, were numerous and large. Stratum granulosum well preserved. In other parts of the cortex nothing noticeably abnormal was found except that the large cells of the third layer seemed rather less numerous than usual.

Case 20.—T. F., male, aged 24, died March 12, 1896, in Danvers Lunatic Hospital, of gangrene of lung. He is said to have had a few fits at the age of fifteen months. They then ceased till he was ten years old; since that time they have been frequent, often several in a day. He was much demented and talked with difficulty.

At the autopsy, the only morbid change noted in the brain was marked shrinking and hardening of both cornua ammonis.

Microscopically, both cornua ammonis presented practically identical lesions. Almost complete atrophy of the stratum pyramidale, a few irregularly disposed cells re-

maining in the usual situation, just before its entrance into the nucleus fasciæ dentatæ, which, on both sides, was almost entirely destitute of nerve-cells. Stratum granulosum thinned in spots; numerous nuclei, but no large spider cells in the atrophied parts. Stained by Nissl's method, the remaining cells in these portions seemed normal. By Pal's method, there was great thinning of the nerve-fibres of the deeper layer of the alveus, and of the fibres radiating into the fascia dentata. The remaining cortex appeared normal.

In addition to the foregoing cases, I met with two in which there was a perceptible difference in the size of the cornua ammonis without any decided difference in consistency, in which microscopical examination showed partial atrophy of the nucleus fasciæ dentatæ, and one, apparently normal to the naked eye, in which there was slight change of the same sort.

In considering the relations of this lesion to epilepsy, the first question is, of course, whether it occurs in other than epileptics, and, if so, how often. So far as my own experience is concerned, I have never found, on the post-mortem table, anything which could be mistaken for the condition I have described in any other than epileptics. Since my attention has been turned to the subject, I have systematically inspected the brains which I have examined with reference to this point. The number so investigated at Little Rock I cannot state precisely, but it was certainly above a hundred. During my service at Danvers I have examined fifty-three brains, or, excluding eight epileptics, forty-five.

I have examined the cornua ammonis of these cases microscopically, with the exception of a few still awaiting examination. In two cases of senile dementia, I found similar changes to those described in a slight degree. In one, a portion of the stratum pyramidale, in the other, a small part of the nucleus fasciæ dentatæ was atrophied. The lesion was not, in either case, enough to make any noticeable difference in the size or consistency of the convolution.

Nerander is the only writer on the subject, so far as I have found, who gives any definite statement of having frequently found induration of the cornu ammonis in other than epileptics, and from the abstract of his paper, it seems probable that the lesions in his cases were other

than those I have described. The facts which I have found in my investigation of this subject may be recapitulated as follows:

1. In nearly 50 per cent. of epileptics examined with reference to this point, I have found one or both of the cornua ammonis smaller and firmer than normal.

2. I have failed to find this lesion in any other cases.

3. In a large proportion of these cases, this condition was associated with other abnormalities of the cerebrum, most frequently microgyria of an entire hemisphere.

4. Whether occurring alone, or associated with other morbid conditions, the histological changes were practically identical in all my cases, consisting in destruction of the neurons having their origin in the stratum pyramidale and nucleus fasciæ dentatæ.

Whatever may be thought of the relation between the lesion and the disease with which it is associated, there seem to me singular and interesting facts. Do they warrant any conclusion as to a relation of cause and effect? In view of the frequency of this condition in epileptics, and what I must consider its great rarity in those not subject to this disease, it seems to me that the question of mere coincidence hardly requires discussion. The dilemma is practically presented, whether the epilepsy causes the anatomical changes or they the epilepsy.

As has been seen, the weight of authority is in favor of the former opinion. At the same time, the principle reason for this appears to be rather the improbability from anything we know of the functions of this convolution, that it can have any special relation to epilepsy, than any definite theory as to the way in which epilepsy could bring about such changes in a single convolution. The only attempt at an explanation that I have seen, that of Wundt, already quoted, assumes an entirely hypothetical increase of intraventricular pressure, and offers no explanation of why it should be, in most cases unilateral, or how it could bring about such a condition if it existed. So far as I am aware, no such result has been reported in cases of hydrocephalus, in which intra-ventricular pressure is known to be excessive.

There remains the supposition that the condition of the cornu ammonis is the cause of the convulsions, which does not, to my mind, present any difficulty. We have to do, practically, with a cicatrix involving the whole cortex

of a pretty large convolution. Two questions arise in this connection: (1) Can a cicatrix of the cerebral cortex cause convulsions? and, (2) Is this liability limited in any such way as to exclude the convolution in question? That irritations of the cortex may cause convulsions is not, I suppose, questioned at the present day. There is abundant evidence, both clinical and experimental, that such may be the case. That a cicatrix of the cortex may act as a focus of irritation is also, I suppose, pretty well made out. I myself have had a case of Jacksonian epilepsy in which the lesion found was an atrophied state of the cortex in the upper part of the fissure of Rolando, histologically very similar to that found in these cases. There remains, then, the question whether an irritation of this particular convolution can be the cause of convulsion. It is not probable that cases often occur in which lesions, other than the one under discussion, are strictly confined to the cornu ammonis. I have met with but one of this description in my reading. (Meynert's 17th case).

"Brain of an epileptic; personal data wanting. Cysticercus of choroid plexus of left cornu ammonis, which is, as it were, soldered to the alveus of the cornu ammonis in an extent of more than a centimetre, which spot appears infiltrated and surrounded by a thick injection of blood-vessels."

Cases are not lacking, however, in which convulsions have been caused by lesions of the temporal lobe.

In case No. 74 of Sommer's collection, it is stated that epileptic attacks and insanity followed an acute cerebral disease. Convulsions usually nocturnal. A medullary cancer, originating in the anterior portion of the right cornu ammonis, has invaded the posterior and middle lobes of the cerebrum.

Bowerman reports¹² a case in which a fracture of the skull from a blow behind the right ear, at the age of fourteen, was followed by chronic epilepsy, beginning four years after the lesion. Post-mortem, a depression of temporal bone was found, with a corresponding depressed cicatrix about two inches in diameter, involving the superior and middle temporo-sphenoidal convolutions.

I myself saw a case, at the Arkansas asylum, of what appeared to be epilepsy, lasting about four years, without

¹²N. Y. State Hospitals Bulletin, Vol. I., p. 385.

any other symptoms except some headache calculated to excite suspicion of any organic lesion, in which, at the autopsy, a psammoma was found, about two inches in horizontal by one-half in vertical diameter, growing from the dura at the tip of the left temporal lobe, which was softened in the greater part of its extent.

There seems, then, to be positive evidence, if it is needed, that irritation of the temporal lobe may excite convulsions. Such being the case, it seems to me most reasonable to conclude that the scar in the cornu ammonis is, in these cases, a focus of irritation which gives rise to the convulsions. I do not suppose that this convolution has any special prerogative in this respect. It seems to me that we are warranted in believing that a scar in any part of the cortex may have such an effect. Certainly, for my own part, if I should find, in a case of epilepsy, a lesion of the same character and extent in any convolution I should have no hesitation in accepting it as a sufficient cause for the convulsions. In my cases in which this convolution has been associated with others in morbid processes, I do not attribute to it any more influence than to the others in causing convulsions. But where, as in a number of my cases, no other lesion can be discovered, it seems to me entirely justifiable to accept the changes in the cornu ammonis as the starting point of the epilepsy.

Whether as cause or effect of the symptoms with which it is associated, it is evident that this convolution is peculiarly subject for some reason to a very definite disease. If the pathology of this condition could be determined, it would, doubtless, go far to settle the question of its relation with unilateral atrophy or arrested development of the brain, in cases of infantile cerebral palsy. If it is, in such cases, to be considered only a part of the general disease of the hemisphere, there would seem to be nothing improbable in supposing that the same cause, whatever it may be, which in one case affects the entire half of the brain, may, in another, act only on one or a few convolutions. The symptoms would, of course, vary with the extent and situation of the disease, and if it were situated outside of the motor area, it might betray itself only by convulsions. In view of the identity of the lesion of the cornu ammonis in my cases of unilateral cerebral atrophy with those in which the disease was confined to the cornu, I cannot help strongly suspecting that there may be a like similarity in the causes.

Unfortunately, the etiology of unilateral atrophy is obscure. Of twelve cases collected by Richardiere,¹³ the disease began in seven with convulsions followed by hemiplegia. In five, paralysis occurred, either congenitally or in infancy, and there is no history of convulsions at the time. For the convulsions themselves, no cause is assigned, with the exception of one case, in which they are said to have followed measles.

Neuroth¹⁴ reports five cases of infantile cerebral paralysis following whooping cough, which he believes to be due to hæmorrhagic encephalitis. In view of the fact that such paralysis often follows infectious diseases, Strumpell's hypothesis of a polio-encephalitis seems as probable as any, but it must await confirmation by autopsies on recent cases. The probability is that children not infrequently die either during or soon after the onset of paralysis, and it is very desirable that such cases should be thoroughly examined. Of this, naturally, there is no opportunity in a hospital for the insane.

Supposing it to be the fact that sclerosis of the cornu ammonis may be a cause of epilepsy, a practical conclusion may be drawn from it. A large proportion of what appear, clinically, to be "essential" epilepsies, are upon this supposition of organic origin. This would be sufficient to account for their incurability, and to render it improbable that any drug will ever be discovered which will permanently cure the symptoms of a lesion in its nature irremediable.

Whatever may be thought of the correctness of the conclusions to which I have been provisionally led, the frequency of the lesion, in connection with epilepsy, and the uniformity and definiteness of its localization, are, it seems to me, matters of great interest, and worthy of further study with a view to the elucidation of the relations, whatever they may be, between it and the associated symptoms.

In conclusion, I wish to make it clear that I do not believe that this convolution has any more to do with epilepsy than any other, apart from the evident fact that it is peculiarly liable to disease. Neither would I be understood as asserting that all epilepsies originate in any part

¹³Etude sur les Scleroses Encephaliques Primitives de l'Enfance, Paris, 1885.

¹⁴Wiener Med. Wochenschrift, 1896, No. 16.

of the cerebral cortex. It seems certain that epileptiform convulsions may be due to peripheral irritations, and to toxæmic conditions, and epilepsy, as we know it, consists in the repetition of such convulsions. I think it altogether probable that many epilepsies depend on such causes. But it does not seem to me necessary to assume, without evidence, such a cause, or to cover our ignorance by saying that epilepsy is a functional disease, in cases in which there is an entirely different anatomical explanation for the symptoms.

DESCRIPTION OF THE DRAWINGS.

Fig. I.—Left cornu ammonis of case 14. Carmine stain. Apparently normal, except for a narrow band of sclerosed tissue, extending from x to y.

(fi) fimbria.

(stpy) stratum pyramidale.

(nfd) nucleus fasciæ dentatæ.

(stgr) stratum granulosum.

Fig. II.—Right cornu ammonis of same case. Sclerosis of stratum pyramidale and nucleus fasciæ dentatæ, with atrophy of their nerve-cells and of the stratum granulosum. References as above.

Fig. III.—Left cornu ammonis, same case. Pal's stain. Normal, or nearly so.

(nfd) nucleus fasciæ dentatæ.

(hfd) hilum fasciæ dentatæ.

(alv) superficial layer of alveus.

(a) deep layer of alveus.

Fig. IV.—Right cornu ammonis, same case. Absence of medullated fibres of hilum fasciæ dentatæ and deeper layer of alveus. References as above.

The drawings for the first three figures are by Dr. Ira Van Gieson, to whom I desire to return my hearty thanks.

ADDENDUM.

Since this article was in print I have found, in both cornua ammonis of a patient dying, in the Danvers Lunatic Hospital, of general paresis, changes identical with those above described. There was no history of epilepsy and the patient did not suffer from convulsions during his stay in the hospital. This would seem, therefore, to be an instance of the occurrence of this lesion independently of convulsions. Judging from the microscopical appearances, I incline to think it of older date than the general paresis.